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NEWS 15 DEC 30 CAPLUS - PATENT COVERAGE EXPANDED  
NEWS 16 JAN 03 No connect-hour charges in EPFULL during January and  
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NEWS 17 JAN 11 CA/CAPLUS - Expanded patent coverage to include Russia  
(Federal Institute of Industrial Property)  
  
NEWS EXPRESS JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT  
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FILE 'HOME' ENTERED AT 16:18:34 ON 21 JAN 2005

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COST IN U.S. DOLLARS	SINCE FILE	TOTAL
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=> s hypercalciuria  
L1 7830 HYPERCALCIURIA

=> s l1 and risk  
L2 1061 L1 AND RISK

=> s hypercalciuria and (increase risk or predisposition or risk factor or disease risk?)

6 FILES SEARCHED...  
L3 342 HYPERCALCIURIA AND (INCREASE RISK OR PREDISPOSITION OR RISK FACTOR OR DISEASE RISK?)

=> d l2 and increase risk  
'AND' IS NOT A VALID FORMAT  
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=> s l2 and increase risk  
L4 1 L2 AND INCREASE RISK

=> d l4 ti abs ibib tot

L4 ANSWER 1 OF 1 USPATFULL on STN  
TI Rapid identification of conditions, compounds, or compositions that inhibit, prevent, induce, modify, or reverse transitions of physical state  
AB The invention concerns arrays comprising hundreds, thousands, to hundreds of thousands of samples and methods for screening thereof. These methods are useful to optimize, select, and discover compounds, compositions, or conditions that prevent, inhibit, induce, modify, or reverse physical-state transitions, particularly in-vivo physical-state

transitions relating to disease causing processes. Such compounds, compositions, or conditions can be exploited to treat (e.g., reverse) or prevent the disease itself, the cause of the disease, or the symptoms of the disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2002:185606 USPATFULL

TITLE: Rapid identification of conditions, compounds, or compositions that inhibit, prevent, induce, modify, or reverse transitions of physical state

INVENTOR(S): Levinson, Douglas, Sherborn, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002098518	A1	20020725
APPLICATION INFO.:	US 2001-994585	A1	20011127 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-253629P	20001128 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	PENNIE AND EDMONDS, 1155 AVENUE OF THE AMERICAS, NEW YORK, NY, 100362711	
NUMBER OF CLAIMS:	80	
EXEMPLARY CLAIM:	1	
LINE COUNT:	3080	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

(FILE 'HOME' ENTERED AT 16:18:34 ON 21 JAN 2005)

FILE 'MEDLINE, USPATFULL, DGENE, FSTA, BIOSIS, WPIDS, EMBASE, BIOTECHDS'  
ENTERED AT 16:19:15 ON 21 JAN 2005

L1 7830 S HYPERCALCIURIA  
L2 1061 S L1 AND RISK  
L3 342 S HYPERCALCIURIA AND (INCREASE RISK OR PREDISPOSITION OR RISK F  
L4 1 S L2 AND INCREASE RISK

=> s l3 and osteoporosis  
L5 66 L3 AND OSTEOPOROSIS

=> s l5 and postmenopause  
L6 15 L5 AND POSTMENOPAUSE

=> d l6 ti abs ibib tot

L6 ANSWER 1 OF 15 MEDLINE on STN  
TI Review of risk factors for **osteoporosis** with particular reference to a possible aetiological role of dietary salt.  
AB Laboratory animal, clinical and epidemiological studies in the published literature have been reviewed in order to establish whether excessive salt intake is an important **risk factor** for the development of **osteoporosis** and whether an intervention strategy based on salt restriction would be beneficial in the prevention of **osteoporosis**. Genetic factors appear to be far more important than the combination of nutritional, hormonal, environmental and lifestyle factors in the pathogenesis of **osteoporosis**. The most important single non-genetic factor is oestrogen deficiency in postmenopausal women. Preventive measures should be aimed at maximizing peak bone mass at skeletal maturity and retarding bone loss thereafter. Apart from postmenopausal oestrogen deficiency, various factors have been

incriminated as risk factors for **osteoporosis**, and these include age at menarche, age at and years since menopause, insufficient physical exercise, alcohol, smoking, low calcium intake, low or high protein intake and high intake of phosphorus, sodium or caffeine. Many of the risk factors are considered to be weak, although when combined they could impact significantly on bone health. Increased intakes of various nutritional factors (potassium, magnesium, zinc, vitamin C), fibre and alkaline-producing fruit and vegetables favour adult bone health. Calcium homeostasis is normally well regulated such that increased calcium loss via the urine leads to increased calcium absorption from the gut. However, the duration of this adaptive process may be greater than that of many of the studies demonstrating that increased salt intake leads to both increased sodium and calcium in the urine. In any case, higher urinary calcium output appears to be seen only in a minority of humans in response to increased salt intake. As numerous factors-genetic, nutritional, hormonal and lifestyle-are involved in the maintenance of calcium homeostasis, it is difficult to devise human studies which adequately take into account all the important factors. Another difficulty is that many past studies have relied on imprecise methods for the measurement of bone resorption. Nor have studies based on the use of the laboratory rat produced clear answers to the problem because the rat, as a species, is uniquely deficient in its ability to handle the relevant minerals. Limited studies to date indicate that increased sodium intake neither exerts a consistent effect on various biomarkers of bone health nor leads to irreversible changes in the bone modelling process in men or in pre- or postmenopausal women. We conclude from the available evidence that increased sodium (or salt) intake is not an important **risk factor for osteoporosis** and that a reduction of salt intake from 9 to 6g/day in the diet would not be beneficial as an intervention measure in the prevention of **osteoporosis**. More research is needed to (i) assess the effects (especially long-term) of various nutrients including sodium on bone health, (ii) assess the long-term value of any intervention strategy involving reduced intake of a particular nutrient such as sodium; and (iii) determine whether subpopulations exist particularly in the elderly (e.g. sodium-responsive subjects) in which adaptation to sodium-induced **hypercalciuria** may be compromised. General prudence dictates that excessively high levels of dietary salt should be eschewed by those persons with raised blood pressure or a limited range of genetic disorders. However, for the generally healthy person there is no sound evidence that the consumption of salt at the present average level of 9g/day constitutes a **risk factor for osteoporosis**.

ACCESSION NUMBER: 2000183656 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 10717363  
TITLE: Review of risk factors for **osteoporosis** with particular reference to a possible aetiological role of dietary salt.  
AUTHOR: Cohen A J; Roe F J  
CORPORATE SOURCE: Toxicology Advisory Services, Hamilton House, 17 Cedar Road, Sutton, Surrey, SM2 5DA, UK.  
SOURCE: Food and chemical toxicology : an international journal published for the British Industrial Biological Research Association, (2000 Feb-Mar) 38 (2-3) 237-53. Ref: 123  
Journal code: 8207483. ISSN: 0278-6915.  
PUB. COUNTRY: ENGLAND: United Kingdom  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
(REVIEW, TUTORIAL)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200005  
ENTRY DATE: Entered STN: 20000518  
Last Updated on STN: 20000518  
Entered Medline: 20000509

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TI Subchondral fractures of the femoral head: A review of seven cases.

AB Objective. - To describe the main characteristics of subchondral fractures of the femoral head. Case-Reports. - The seven patients, five women and two men, with a mean age of 50 years (37-76 years), presented with mechanical pain in the groin. Bone loss was the main **risk factor**. Two patients had postmenopausal **osteoporosis** (including one with a history of ovariectomy at 30 years of age), two had **osteoporosis** induced by glucocorticoid therapy given after transplantation (liver and allogeneic bone marrow, respectively), one had an ACTH-producing adenoma, and one had femoral osteopenia at a site of topical glucocorticoid therapy for atopic dermatitis. The remaining patient had osteopenia and a history of smoking. Phosphate and calcium levels were normal in five patients. One patient had isolated hypocalciuria and another had moderate proximal tubular disease with phosphate wasting and **hypercalciuria**. Magnetic resonance imaging (MRI) disclosed a subcapital line of low signal on T1- and T2-weighted sequences surrounded by an area of variable size generating low signal on T1 images and high signal on T2 images, with postgadolinium enhancement, denoting marrow edema. Complete elimination of weight bearing for 6 weeks, symptomatic agents, and treatment of the underlying causes of bone insufficiency were used in all seven patients. Mean follow-up was 2.4 years (range, 11-39 years). No cases of osteonecrosis were recorded. Conclusion. - Several cases of subchondral fracture have been reported in the literature. Bone insufficiency was the main **risk factor** in all the patients. .COPYRGHT. 2004 Elsevier SAS. All rights reserved.

ACCESSION NUMBER: 2004145602 EMBASE

TITLE: Subchondral fractures of the femoral head: A review of seven cases.

AUTHOR: Gerot I.L.; Demondion X.; Louville A.B.; Delcambre B.; Cortet B.

CORPORATE SOURCE: I.L. Gerot, Rheumatology Department, Lille Teaching Hospital, 59037 Lille, France. igerot@chru-lille.fr

SOURCE: Joint Bone Spine, (2004) 71/2 (131-135).  
Refs: 19  
ISSN: 1297-319X CODEN: JBSPFA

PUBLISHER IDENT.: S 1297-319X(03)00050-2

COUNTRY: France

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 005 General Pathology and Pathological Anatomy  
033 Orthopedic Surgery  
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

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TI Pharmacotherapeutics for **osteoporosis** prevention and treatment.

AB **Osteoporosis** is a silent disease that affects 10 million Americans; 80% of those affected are women. Although the disease is more common in postmenopausal Caucasian women, all ages and races are at risk. **Osteoporosis** can be a debilitating disease that can cause pain, fractures, depression, and social withdrawal. Signs of **osteoporosis** include kyphosis, loss of height, and protrusion of the abdomen. Because symptoms generally do not occur until after the disease has progressed, clinicians should include **osteoporosis** screening and preventative education as part of the regular gynecologic care. Diagnosis is typically made by a dual energy x-ray absorptiometry (DEXA) scan. Treatment consists of dietary and lifestyle changes, along with pharmacologic intervention. Although hormone therapy has been shown to be effective in preventing **osteoporosis**, the risks of

long-term treatment with HRT are discussed. The following effective treatment options for women who have been diagnosed with the disease are discussed: bisphosphonates, calcitonin, and selective estrogen receptor modulators (SERMs). Because midwives regularly care for women of all ages, they are ideal candidates to provide women with preventative education, screening, counseling, and treatment. .COPYRGT. 2003 American College of Nurse-Midwives.

ACCESSION NUMBER: 2004141618 EMBASE  
TITLE: Pharmacotherapeutics for **osteoporosis** prevention and treatment.  
AUTHOR: Davidson M.R.  
CORPORATE SOURCE: Dr. M.R. Davidson, 44108 Bristow Circle, Ashburn, VA 20147, United States  
SOURCE: Journal of Midwifery and Women's Health, (2003) 48/1 (39-52).  
Refs: 49  
ISSN: 1526-9523 CODEN: JMWHAA  
PUBLISHER IDENT.: S 1526-9523(02)00359-8  
COUNTRY: United States  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 010 Obstetrics and Gynecology  
017 Public Health, Social Medicine and Epidemiology  
033 Orthopedic Surgery  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
SUMMARY LANGUAGE: English

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TI Prevention and treatment of corticosteroid-induced **osteoporosis** in the elderly.

AB Corticosteroid-induced **osteoporosis** is a serious complication of corticosteroid therapy. Bone loss occurs within the first three to six months of corticosteroid use. Fracture rates can be quite high within the first year of corticosteroid therapy. All patients on or starting corticosteroids need to have assessments for secondary causes of **osteoporosis**, counseling for modification of risk factors, and measures taken to prevent fractures. All patients should have a calcium intake of 1200-1500 mg per day and vitamin D supplementation of 800-1000 IU per day. Bisphosphonates are currently the most potent antiresorptive agent. This class of medication has also been shown to reduce the occurrence of vertebral fractures. Bisphosphonate therapy is the recommended first-line agent both for the prevention and treatment of corticosteroid-induced **osteoporosis**.

ACCESSION NUMBER: 2003177353 EMBASE  
TITLE: Prevention and treatment of corticosteroid-induced **osteoporosis** in the elderly.  
AUTHOR: Boulos P.; Papaioannou A.; Adachi J.D.  
CORPORATE SOURCE: Dr. J.D. Adachi, 501-25 Charlton Ave E, Hamilton, Ont. L8N 1Y2, Canada. jd.adachi@sympatico.ca  
SOURCE: Annals of Long-Term Care, (1 Jan 2003) 11/1 (42-48).  
Refs: 51  
ISSN: 1524-7929 CODEN: ALTCHF  
COUNTRY: United States  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 003 Endocrinology  
020 Gerontology and Geriatrics  
033 Orthopedic Surgery  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
SUMMARY LANGUAGE: English

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TI Clinical evaluation for **osteoporosis**.

AB The clinical evaluation of the osteoporotic patient should include a careful assessment of risk factors for low bone mass, falls, and fractures; quantitation of BMD; a thorough medical history and physical examination; and a targeted set of laboratory, radiographic, and other diagnostic studies as indicated. Among the elderly, vitamin D deficiency ranks high as one of the most underdiagnosed and yet reversible causes of **osteoporosis**. Regardless of age, every patient with low bone mass or fractures deserves an evaluation to uncover reversible, treatable disorders and to detect serious underlying illnesses.

ACCESSION NUMBER: 2003174526 EMBASE

TITLE: Clinical evaluation for **osteoporosis**.

AUTHOR: Becker C.

CORPORATE SOURCE: Dr. C. Becker, Toni Stabile Osteoporosis Center, Columbia Presbyterian Medical Center, Harkness Pavilion-904, 180 Fort Washington Avenue, New York, NY 10032, United States. cb2006@columbia.edu

SOURCE: Clinics in Geriatric Medicine, (2003) 19/2 (299-320).  
Refs: 137  
ISSN: 0749-0690 CODEN: CGMEE6

PUBLISHER IDENT.: S 0749-0690(02)00068-X

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 003 Endocrinology  
017 Public Health, Social Medicine and Epidemiology  
020 Gerontology and Geriatrics  
037 Drug Literature Index  
038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

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TI **Osteoporosis** - An overview.

AB **Osteoporosis** is a systemic progressive disease with important clinical complications because of the fractures that arise and cause morbidity in especially the aging postmenopausal women. It is recognized as an important public health problem because of the significant morbidity and mortality associated with its complication, namely fractures of the proximal femur (hip), vertebrae (spine), distal forearm, proximal humerus, pelvis, and other skeletal sites. Compared with other osteoporotic fractures, however, fractures of the hip incur the greatest morbidity and direct medical costs for health services. There are now a variety of treatments available for the management of **osteoporosis**. The inhibitors of bone resorption, which include calcium, the vitamin Ds, bisphosphonates, calcitonins and gonadal steroids have been variously shown to prevent bone loss or to reduce fractures. On the other hand bone formation stimulating agents as fluorides must be considered also. However, prevention of **osteoporosis** during the teen and early adult years is far superior to any of the treatment for older individuals with identification of risk factors, careful examination and a few simple diagnostic tests. The purpose of this review is to provide an general overview of an **osteoporosis**.

ACCESSION NUMBER: 2002180824 EMBASE

TITLE: **Osteoporosis** - An overview.

AUTHOR: Iqbal M.M.; Mahmud S.Z.

CORPORATE SOURCE: M.M. Iqbal, Department of Epidemiology, School of Public Health, University of Alabama at Birmingham, Birmingham, AL, United States

SOURCE: Bangladesh Journal of Obstetrics and Gynecology, (2001) 16/1 (27-34).  
Refs: 20

ISSN: 1018-4287 CODEN: BJOGFX  
COUNTRY: Bangladesh  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 010 Obstetrics and Gynecology  
017 Public Health, Social Medicine and Epidemiology  
033 Orthopedic Surgery  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
SUMMARY LANGUAGE: English

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TI Complementary therapies for reducing the risk of **osteoporosis** in patients receiving luteinizing hormone-releasing hormone treatment/orchiectomy for prostate cancer: A review and assessment of the need for more research.

AB **Osteoporosis** in women has received a substantial amount of attention, but its impact in men is also significant and noteworthy. Those men who benefit from treatment for prostate cancer with androgen deprivation therapy (ADT) may also be at a higher risk for **osteoporosis**. Pharmacologic approaches to reduce this risk have received some attention. For example, agents such as bisphosphonates, estrogen receptor-binding drugs (diethylstilbestrol, tamoxifen, and raloxifene), calcitonin, and fluoride are some of the more promising interventions that have been previously outlined. In addition, statin drugs, or hepatic 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors, have recently been hypothesized to lower **osteoporosis** risk. However, complementary therapies, which may also have an impact on reducing **osteoporosis** risk, have not received attention. Dietary and supplemental calcium and vitamin D have been shown, in some preliminary investigations, to maintain bone density in women and men. Numerous healthy and affordable dietary sources of this mineral and vitamin exist, and large intakes can be realistically achieved through proper education. Similarly, the supplemental dosages required to impact risk have been moderate, appear to be safe, are of low cost, and thus may provide an additional route for reducing risk, especially if these interventions are initiated at the start of medical treatment. More studies in men receiving ADT are needed because the existing work has mostly focused on men without castrate levels of male hormone. Additionally, many studies with conventional and nonconventional agents have only focused on individuals with baseline **osteoporosis**, rather than normal bone mineral densities or osteopenia. Other promising complementary therapies, such as weight-bearing exercise and abstaining from smoking, may also be of benefit. Newer estrogenic-type supplements (eg, ipriflavone) appear interesting and have some preliminary data, but more research is desperately required to determine their actual impact and potential for adverse effects (such as lymphocytopenia from a recent trial). Simple, inexpensive, and potentially effective dietary and supplemental approaches to reduce the risk of **osteoporosis** in men exist, and they should be discussed with patients. Whether these approaches effectively reduce the risk of **osteoporosis** in men receiving androgen ablation remains to be determined. The possibility is intriguing, and future research is needed. In the meantime, it is important to keep in mind that these complementary approaches are, at the very least, an integral part of the conventional options used today to the reduce the risk of **osteoporosis** in men and women. .COPYRGT. 2002 Elsevier Science Inc.

ACCESSION NUMBER: 2002131130 EMBASE

TITLE: Complementary therapies for reducing the risk of **osteoporosis** in patients receiving luteinizing hormone-releasing hormone treatment/orchiectomy for prostate cancer: A review and assessment of the need for more research.



AUTHOR: Moyad M.A.  
 CORPORATE SOURCE: M.A. Moyad, Department of Urology, University of Michigan Med. Center, 1500 East Medical Center Drive, Ann Arbor, MI 48109-0330, United States. moyad@umich.edu  
 SOURCE: Urology, (2002) 59/4 SUPPL. 1 (34-40).  
 Refs: 58  
 ISSN: 0090-4295 CODEN: URGYAZ  
 PUBLISHER IDENT.: S 0090-4295(01)01174-8  
 COUNTRY: United States  
 DOCUMENT TYPE: Journal; General Review  
 FILE SEGMENT: 003 Endocrinology  
 016 Cancer  
 028 Urology and Nephrology  
 037 Drug Literature Index  
 038 Adverse Reactions Titles  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English

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TI American association of clinical endocrinologists 2001 medical guidelines for clinical practice for the prevention and management of postmenopausal **osteoporosis**.

ACCESSION NUMBER: 2001272656 EMBASE  
 TITLE: American association of clinical endocrinologists 2001 medical guidelines for clinical practice for the prevention and management of postmenopausal **osteoporosis**.

SOURCE: Endocrine Practice, (2001) 7/4 (294-312).  
 Refs: 87

ISSN: 1530-891X CODEN: EPNRAT

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 003 Endocrinology  
 010 Obstetrics and Gynecology  
 014 Radiology  
 033 Orthopedic Surgery  
 037 Drug Literature Index  
 038 Adverse Reactions Titles

LANGUAGE: English

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TI **Osteoporosis** in women with spinal cord injuries.

AB Decreased bone density and increased fracture risk are seen in patients with SCI. The bone resorption rate is markedly increased. **Hypercalciuria**, low PTH, and low 1,25 (OH)(2) vitamin D are commonly seen. Bed-rest studies show similar findings, but of lesser magnitude. Therapies to treat or prevent **osteoporosis** include optimal nutrition (with care to avoid exacerbating **hypercalciuria**). Weight-bearing or functional electrical stimulation cycle ergometry may prevent some of the bone loss, especially in acutely injured patients. Estrogen should be considered in postmenopausal or amenorrheic women, but not if they are at high risk of thromboembolism. More research on effects of estrogen is needed in this population. Bisphosphonates may also help prevent the acute bone loss; oral routes must not be used in recumbent patients. Thiazides could be useful as adjunct therapy.

ACCESSION NUMBER: 2001031537 EMBASE

TITLE: **Osteoporosis** in women with spinal cord injuries.

AUTHOR: Ott S.M.

CORPORATE SOURCE: Dr. S.M. Ott, Division of Metabolism, University of Washington, 1959 NE Pacific Street, Seattle, WA 98195-6426, United States

SOURCE: Physical Medicine and Rehabilitation Clinics of North America, (2001) 12/1 (111-131).

Refs: 64  
 ISSN: 1047-9651 CODEN: PMRAFZ  
 COUNTRY: United States  
 DOCUMENT TYPE: Journal; General Review  
 FILE SEGMENT: 019 Rehabilitation and Physical Medicine  
 031 Arthritis and Rheumatism  
 033 Orthopedic Surgery  
 037 Drug Literature Index  
 038 Adverse Reactions Titles  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English

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TI Risk of calcium oxalate nephrolithiasis after calcium or combined calcium and calcitriol supplementation in postmenopausal women.

AB Although calcium supplementation can cause **hypercalciuria**, the risk of nephrolithiasis has been shown to decrease rather than increase among subjects who had a higher calcium intake. **Hypercalciuria** is also a well-established side effect of calcitriol administration. However, the risk of nephrolithiasis is not well defined. The present study was undertaken to prospectively determine the effect of calcium with or without calcitriol on physicochemical risk factors associated with calcium oxalate nephrolithiasis in Thai postmenopausal women with **osteoporosis**. Subjects consisted of 53 Thai women more than 10 years postmenopausal who were randomly allocated to receive 750 mg of calcium carbonate supplement alone (n = 28) or 750 mg of calcium carbonate plus 0.5 µg calcitriol (n = 25) daily. Mean ± SEM for age was 65.3 ± 1.1 years, body weight 53.5 ± 1.3 kg. Urine samples for biochemical assays were collected at baseline and 3 months after treatment. Supersaturation for calcium oxalate stone formation was assessed from the 24 h urine constituents by the Tiselius's index, AP(CaOx). Three months of calcium supplement alone resulted in a modest, but not significant, increase in urinary calcium (baseline, 2.90 ± 0.43 mmol/day; after treatment 3.58 ± 0.54 mmol/day) with no change in urinary oxalate, citrate or magnesium. In contrast, calcium together with calcitriol caused a significant increase in urinary calcium (baseline, 2.87 ± 0.41 mmol/day; after treatment, 4.08 ± 0.57 mmol/day; p < 0.05). No significant change in other urine constituents after treatment with calcium and calcitriol was detected. Therefore, AP(CaOx) did not significantly increase either after calcium alone (baseline, 1.17 ± 0.39; after treatment, 1.36 ± 0.28) or after calcium plus calcitriol (baseline, 1.09 ± 0.17; after treatment, 1.09 ± 0.19). However, after treatments, 12 subjects (23%) - 6 receiving calcium supplement alone and 6 receiving calcium plus calcitriol supplement - had high AP(CaOx) values (greater than the upper limit of 95% CI for AP(CaOx) derived from non-stone-forming Thai women). The post-treatment/baseline ratio was 3.21 ± 0.74 for urinary calcium, 1.01 ± 0.19 for urinary oxalate, and 2.23 ± 0.42 (median 1.15) for AP(CaOx). The post-treatment/baseline ratio of calcium, but not for urinary oxalate, had a significant correlation with the post-treatment/baseline ratio of AP(CaOx). Our findings suggest that the alteration in the risk of calcium oxalate nephrolithiasis based on urinary composition is related to the alteration in urinary calcium. The risk of calcium oxalate nephrolithiasis does not increase significantly after calcium or combined calcium and calcitriol supplement in the majority of postmenopausal women with **osteoporosis**.

ACCESSION NUMBER: 2000318421 EMBASE

TITLE: Risk of calcium oxalate nephrolithiasis after calcium or combined calcium and calcitriol supplementation in postmenopausal women.

AUTHOR: Domrongkitchaiporn S.; Ongphiphadhanakul B.; Stichtantrakul W.; Piaseu N.; Chansirikarn S.; Puavilai G.; Rajatanavin R.

CORPORATE SOURCE: Dr. S. Domrongkitchaiporn, Department of Medicine,

Ramathibodi Hospital, Rama 6, Bangkok 10400, Thailand.  
 rasdr@mahidol.ac.th  
 SOURCE: Osteoporosis International, (2000) 11/6 (486-492).  
 Refs: 37  
 ISSN: 0937-941X CODEN: OSINEP  
 COUNTRY: United Kingdom  
 DOCUMENT TYPE: Journal; Article  
 FILE SEGMENT: 028 Urology and Nephrology  
 033 Orthopedic Surgery  
 037 Drug Literature Index  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English

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 on STN

TI Review of risk factors for **osteoporosis** with particular  
 reference to a possible aetiological role of dietary salt.  
 AB Laboratory animal, clinical and epidemiological studies in the published  
 literature have been reviewed in order to establish whether excessive salt  
 intake is an important **risk factor** for the development  
 of **osteoporosis** and whether an intervention strategy based on  
 salt restriction would be beneficial in the prevention of  
**osteoporosis**. Genetic factors appear to be far more important than  
 the combination of nutritional, hormonal, environmental and lifestyle  
 factors in the pathogenesis of **osteoporosis**. The most important  
 single non-genetic factor is oestrogen deficiency in postmenopausal women.  
 Preventive measures should be aimed at maximizing peak bone mass at  
 skeletal maturity and retarding bone loss thereafter. Apart from  
 postmenopausal oestrogen deficiency, various factors have been  
 incriminated as risk factors for **osteoporosis**, and these include  
 age at menarche, age at and years since menopause, insufficient physical  
 exercise, alcohol, smoking, low calcium intake, low or high protein intake  
 and high intake of phosphorus, sodium or caffeine. Many of the risk  
 factors are considered to be weak, although when combined they could  
 impact significantly on bone health. Increased intakes of various  
 nutritional factors (potassium, magnesium, zinc, vitamin C), fibre and  
 alkaline-producing fruit and vegetables favour adult bone health. Calcium  
 homeostasis is normally well regulated such that increased calcium loss  
 via the urine leads to increased calcium absorption from the gut. However,  
 the duration of this adaptive process may be greater than that of many of  
 the studies demonstrating that increased salt intake leads to both  
 increased sodium and calcium in the urine. In any case, higher urinary  
 calcium output appears to be seen only in a minority of humans in response  
 to increased salt intake. As numerous factors - genetic, nutritional,  
 hormonal and lifestyle - are involved in the maintenance of calcium  
 homeostasis, it is difficult to devise human studies which adequately take  
 into account all the important factors. Another difficulty is that many  
 past studies have relied on imprecise methods for the measurement of bone  
 resorption. Nor have studies based on the use of the laboratory rat  
 produced clear answers to the problem because the rat, as a species, is  
 uniquely deficient in its ability to handle the relevant minerals. Limited  
 studies to date indicate that increased sodium intake neither exerts a  
 consistent effect on various biomarkers of bone health nor leads to  
 irreversible changes in the bone modelling process in men or in pre- or  
 postmenopausal women. We conclude from the available evidence that  
 increased sodium (or salt) intake is not an important **risk**  
**factor** for **osteoporosis** and that a reduction of salt  
 intake from 9 to 6 g/day in the diet would not be beneficial as an  
 intervention measure in the prevention of **osteoporosis**. More  
 research is needed to (i) assess the effects (especially long-term) of  
 various nutrients including sodium on bone health, (ii) assess the  
 long-term value of any intervention strategy involving reduced intake of a  
 particular nutrient such as sodium; and (iii) determine whether  
 subpopulations exist particularly in the elderly (e.g. sodium-responsive

subjects) in which adaptation to sodium-induced **hypercalciuria** may be compromised. General prudence dictates that excessively high levels of dietary salt should be eschewed by those persons with raised blood pressure or a limited range of genetic disorders. However, for the generally healthy person there is no sound evidence that the consumption of salt at the present average level of 9 g/day constitutes a **risk factor for osteoporosis**. (C) 2000 Elsevier Science Ltd.

ACCESSION NUMBER: 2000091904 EMBASE  
TITLE: Review of risk factors for **osteoporosis** with particular reference to a possible aetiological role of dietary salt.  
AUTHOR: Cohen A.J.; Roe F.J.C.  
CORPORATE SOURCE: A.J. Cohen, Toxicology Advisory Services, Hamilton House, 17 Cedar Road, Sutton, Surrey SM2 5AD, United Kingdom  
SOURCE: Food and Chemical Toxicology, (2000) 38/2-3 (237-253).  
Refs: 123  
ISSN: 0278-6915 CODEN: FCTOD7  
PUBLISHER IDENT.: S 0278-6915(99)00145-3  
COUNTRY: United Kingdom  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 029 Clinical Biochemistry  
LANGUAGE: English  
SUMMARY LANGUAGE: English

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TI [**Osteoporosis**].  
OSTEOPOROSE.

ACCESSION NUMBER: 2000027931 EMBASE  
TITLE: [**Osteoporosis**].  
OSTEOPOROSE.  
AUTHOR: Rohart C.; Benhamou C.L.  
CORPORATE SOURCE: Dr. C. Rohart, Service de Rhumatologie, Hopital Porte Madeleine, CHR Orleans, 45032 Orleans Cedex I, France  
SOURCE: Revue du Praticien, (1 Jan 2000) 50/1 (85-92).  
ISSN: 0035-2640 CODEN: REPRA3  
COUNTRY: France  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 006 Internal Medicine  
LANGUAGE: French

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TI **Osteoporosis** and systemic lupus erythematosus: Etiology and treatment strategies.

AB The importance of bone loss in patients with systemic lupus erythematosus can not be over emphasized. Risk factors for **osteoporosis** in these patients include not only those that apply to the general population, but also many that are related to the underlying disease process, or its treatment. Ongoing research should attempt to clarify the prevalence of low bone mass in lupus, as well as the pathogenic mechanisms applying especially to this population. Strategies for the prevention and treatment of bone loss in lupus patients do not differ significantly from those in the general population. Special attention must be given to the prevention of steroid-induced bone loss, as well as to the gonadal effects of cytotoxic agents.

ACCESSION NUMBER: 96283369 EMBASE  
DOCUMENT NUMBER: 1996283369  
TITLE: **Osteoporosis** and systemic lupus erythematosus: Etiology and treatment strategies.  
AUTHOR: Segal L.G.; Lane N.E.  
CORPORATE SOURCE: Division of Rheumatology, Box 0868, University of California, San Francisco, CA 94143, United States  
SOURCE: Annales de Medecine Interne, (1996) 147/4 (281-289).

ISSN: 0003-410X CODEN: AMDIBO  
COUNTRY: France  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 006 Internal Medicine  
031 Arthritis and Rheumatism  
037 Drug Literature Index  
038 Adverse Reactions Titles  
LANGUAGE: English  
SUMMARY LANGUAGE: English; French

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TI Bone and the 'Comforts of Life'.

AB Coffee drinking, smoking and especially alcohol abuse are considered to be risk factors for fractures and **osteoporosis**. Caffeine causes acute increase in urinary calcium excretion, but epidemiological evidence for the effects of coffee consumption on the risk of fractures is contradictory. Many, (but not all) studies point to decreased bone mass or increased fracture risk in smokers. Alcohol abuse is associated with deleterious changes in bone structure detected by histomorphometry, and with a decrease in bone mineral density (BMD). These changes may also be produced by factors commonly associated with alcohol abuse, e.g. nutritional deficiencies, liver damage and hypogonadism. Alcohol, however, has clear-cut direct effects on bone and mineral metabolism. Acute alcohol intoxication causes transitory hypoparathyroidism with resultant hypocalcaemia and **hypercalciuria**. As assessed by serum osteocalcin levels, prolonged moderate drinking decreases the function of osteoblasts, the bone-forming cells. In addition, chronic alcoholics are characterized by low serum levels of vitamin D metabolites. Thus, alcohol seems to have a direct toxic effect on bone and mineral metabolism. In contrast, it has recently been reported that moderate alcohol consumption by postmenopausal women may have a beneficial effect on bone.

ACCESSION NUMBER: 93250294 EMBASE

DOCUMENT NUMBER: 1993250294

TITLE: Bone and the 'Comforts of Life'.

AUTHOR: Laitinen K.; Valimaki M.

CORPORATE SOURCE: Research Unit of Alcohol Diseases, Helsinki University  
Central Hospital, Tukholmankatu 8 F, SF-00290 Helsinki,  
Finland

SOURCE: Annals of Medicine, (1993) 25/4 (413-425).

ISSN: 0785-3890 CODEN: ANMDEU

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 005 General Pathology and Pathological Anatomy  
017 Public Health, Social Medicine and Epidemiology  
033 Orthopedic Surgery

LANGUAGE: English

SUMMARY LANGUAGE: English

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on STN

TI [**Osteoporosis** due to hormone deficiency: Prevention by sex hormone substitution!].

OSTEOPOROSE DURCH ENDOKRINES DEFIZIT-SYNDROM. VERMEIDBAR DURCH SEXUAL-HORMON-SUBSTITUTION!.

AB The postmenopausal withdrawal of estrogens results in a vicious circle, which is initiated by accelerated bone resorption, hypercalcemia, failure of the calcium retaining system ('escape' phenomenon), and hypercalcuria, and is associated with accelerated bone turnover, and a negative balance of calcium and bone metabolism. To maintain homeostasis, the waste of bone calcium has to be continually balanced by additional osteolysis. Bone resorption is inevitably followed by **osteoporosis** after a varying duration. Following intake of estrogens, the vicious circle is interrupted by deceleration of osteolysis and bone turnover, and a

positive balance of calcium and bone metabolism is restored.  
**Osteoporosis**, as a consequence of postmenopausal endocrine deficiency, may therefore be prevented by a substitution with female sex steroids in due time.

ACCESSION NUMBER: 92068750 EMBASE  
DOCUMENT NUMBER: 1992068750  
TITLE: [Osteoporosis due to hormone deficiency:  
Prevention by sex hormone substitution!].  
OSTEOPOROSE DURCH ENDOKRINES DEFIZIT-SYNDROM. VERMEIDBAR  
DURCH SEXUAL-HORMON-SUBSTITUTION!.

AUTHOR: Nocke W.  
CORPORATE SOURCE: Abteilung fur Gynakologische Endokrinologie, Zentrum fur  
Geburtshilfe und Frauenheilkunde, Rheinische  
Friedrich-Wilhelms-Universitat, W-5300 Bonn-Venusberg,  
Germany

SOURCE: Therapiewoche, (1992) 42/7 (350-357).  
ISSN: 0040-5973 CODEN: THEWA6

COUNTRY: Germany  
DOCUMENT TYPE: Journal; General Review  
FILE SEGMENT: 010 Obstetrics and Gynecology  
033 Orthopedic Surgery  
037 Drug Literature Index

LANGUAGE: German  
SUMMARY LANGUAGE: German; English

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(FILE 'HOME' ENTERED AT 16:18:34 ON 21 JAN 2005)

FILE 'MEDLINE, USPATFULL, DGENE, FSTA, BIOSIS, WPIDS, EMBASE, BIOTECHDS'  
ENTERED AT 16:19:15 ON 21 JAN 2005

L1 7830 S HYPERCALCIURIA  
L2 1061 S L1 AND RISK  
L3 342 S HYPERCALCIURIA AND (INCREASE RISK OR PREDISPOSITION OR RISK F  
L4 1 S L2 AND INCREASE RISK  
L5 66 S L3 AND OSTEOPOROSIS  
L6 15 S L5 AND POSTMENOPAUSE

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E1 2 REED Y/AU  
E2 1 REED Z H/AU  
E3 0 --> REED-GITOMER/AU  
E4 1 REEDA A/AU  
E5 1 REEDA C F/AU  
E6 1 REEDAL D C/AU  
E7 1 REEDAL D R/AU  
E8 1 REEDAL DONNA R/AU  
E9 3 REEDAL J S/AU  
E10 1 REEDD J D/AU  
E11 1 REEDDY K/AU  
E12 1 REEDE COOLEY JR N/AU